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Emotional-Processing in Posttraumatic Stress Disorder II: Startle Reflex Modulation During Picture Processing

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This study examined the impact of a trauma-related stressor on subsequent emotional behavior in veterans with (n = 35) and without (n = 24) posttraumatic stress disorder (PTSD). Self-report and physiological responses, including acoustic startle, were recorded during viewing of emotionally evocative photographs at baseline and following exposure to trauma-related and non-trauma-related stressors. The 2 groups exhibited equivalent patterns of emotional response across self-report and physiological measures at baseline. In contrast, following the trauma challenge, participants with PTSD showed a pattern of startle modulation suggestive of greater defensive reactivity and reduced visual perceptual engagement. These findings, along with augmented corrugator EMG reactivity during the same interval, suggest that trauma-related reexperiencing primes subsequent negative emotional responding in individuals with PTSD.

Posttraumatic stress disorder (PTSD) is associated with marked disturbances in affective processes that develop in response to a psychologically traumatic experience. The *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV*; American Psychiatric Association, 1994) definition of the disorder lists several forms of such disturbances including intense distress upon exposure to trauma-related cues and restricted range of affect or *emotional numbing.* The latter refers to feelings of detachment from others, disinterest in normally pleasurable activities, and a deficit in the capacity to experience and express emotions, especially positive emotions associated with intimacy, tenderness, and sexuality.

Theorists have hypothesized that the hyperarousal and numbing symptoms of PTSD are inversely related and characterized by alternating periods of intense re-experiencing and negative arousal followed by intervals of dampened affective responsivity (Herman, 1992; Horowitz, 1986; van der Kolk, 1987; van der Kolk, Greenberg, Boyd, & Krystal, 1985). Litz (1992) also proposed that emotional numbing in PTSD is phasic, but conceptualized the phenomenon as a transient depletion or reduction in the capacity for *positive emotion* which follows in the wake of episodes of intense reexperiencing and trauma-related arousal. In other words, the Litz model posits that affective abnormalities in PTSD are (a) secondary to the activation of trauma-related conditioned emotional responses (CERs) and accompanying hyperarousal and (b) reflected in hyporeactivity to stimuli that normally evoke an hedonic/appetitive response. Litz also proposed that (c) exposure to trauma-related cues primes the aversive emotional system resulting in facilitation of subsequent defensive responses and hyperreactivity to unpleasant stimuli—an hypothesized consequence of the CER that contradicts the notion of numbing involving a generalized reduction in responsivity.

Preliminary support for the first two of these propositions was provided by a recent study that showed that activation of a trauma-related CER produced phasic deficits in the expression of positive affect in individuals with PTSD (Litz, Orsillo, Kaloupek, & Weathers, 2000). In that study, combat veterans with and without PTSD viewed emotionally evocative pictures before and after exposure to a combat-related audiovisual presentation while their self-report and physiological responses, including facial electromyography (EMG), were recorded. Results showed that the two groups exhibited equivalent patterns of affective response prior to the trauma-related challenge, yet after that manipulation participants with PTSD exhibited suppressed zygomatic EMG (i.e., smile) responses during the viewing of pleasant images relative to the responses of control participants. Contrary to Litz's third prediction, however, no support was found for the hypothesis that the activation of a trauma-related CER would prompt greater defensive reactivity in individuals with PTSD. Participants with PTSD were no more or less reactive to negative stimuli than those without the disorder.

One possible explanation for this null result was that the trauma challenge evoked a mood characterized primarily by anhedonia (i.e., depression/low positive affectivity) rather than heightened arousal/negative affectivity (i.e., fear/anxiety). Another was that the measures of negative affectivity used in the study (i.e., corrugator EMG and self-report ratings of the response to each image) were insensitive to changes in defensive reactivity that may have occurred in response to the trauma-related challenge. Thus, a primary objective of the present study was to replicate and extend this work using (a) a challenge designed to elicit greater defensive

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reactivity and (b) a physiological measure that is a more sensitive indicator of such processes—the *acoustic startle reflex.*

Affect-Modulated Startle

Startle reflex methodology offers a potentially useful tool for examining the impact of activation of a trauma-related CER on subsequent emotional behavior for several reasons. First, the eyeblink response to a startle-eliciting "probe" stimulus is reliably potentiated during negative emotional states, whether evoked by viewing unpleasant pictures (Bradley, Cuthbert, & Lang, 1990; Vrana, Spence, & Lang, 1988), imagery of fear or anger-related scenes (Miller, Levenston, & Patrick, 2002), or anticipation of aversive stimuli (Miller, Curtin, & Patrick, 1999; Miller & Patrick, 2000). Blink potentiation is believed to occur as a function of the match/mismatch between the strategic response disposition prompted by the aversive context (i.e., picture, imagery, etc.) and the defensive eyeblink reflex elicited by the intervening noise probe (Lang, Bradley, & Cuthbert, 1990). That is, when the affective state is aversive (i.e., during viewing of unpleasant pictures), there is a match between the ongoing defensive disposition and the response to the startle probe stimulus resulting in potentiation of the protective blink response. Conversely, when the affective state is positive (i.e., during viewing of pleasant pictures) a mismatch occurs and the defensive startle reaction is attenuated (Bradley, Cuthbert, & Lang, 1999).

Second, there is evidence that startle reflex modulation is sensitive to individual differences in the strength of defensive activation and laboratory manipulations of state negative affect. For example, individuals with specific phobias (Hamm, Cuthbert, Globisch, & Vaitl, 1997) and those who are high in trait fearfulness (Cook, 1999) exhibit more pronounced startle potentiation during viewing of unpleasant stimuli compared with control individuals, whereas psychopaths, who are characterized by deficits in fear responding, exhibit an absence of normal startle potentiation (Levenston, Patrick, Bradley, & Lang, 2000; Patrick, Bradley, & Lang, 1993). Also, Miller and Patrick (2000) observed that the startle response during viewing of threatening words was augmented in high trait anxious individuals during conditions involving threat of shock, but not during safe conditions.

Third, startle reflex modulation can also provide an index of the degree of attentional engagement in processing of a visual foreground stimulus. During processing of engaging/interesting photographs, blink responses to acoustic startle probes are attenuated relative to responses elicited during processing of less engaging pictures (e.g., Simons & Zelson, 1985) or during intertrial intervals (ITI) when no visual stimulus is present (e.g., Codispoti, Bradley, & Lang, 2001). Thus, the difference between the amplitude of the acoustic startle response elicited during picture processing compared with ITI provides an index of the degree of perceptual engagement in processing of the foreground stimulus. In light of data suggesting that exposure to trauma-related stimuli may evoke a state of detachment and disengagement in some individuals with PTSD (e.g., Griffin, Resick, & Mechanic, 1997), we examined this index to gauge the impact of a trauma challenge on perceptual engagement to emotionally evocative stimuli.

For these reasons, startle reflex modulation can provide a sensitive tool for the study of PTSD group differences in emotional response and the effects of experimental manipulations (e.g., exposure to a trauma-related challenge) on those processes. Moreover, when used in conjunction with other measures of emotional behavior including heart rate (HR), skin conductance (SC), and facial EMG responses, we expected that it might also shed light on theoretical issues that bear on the understanding of the locus and mechanisms of emotional-processing abnormalities in PTSD.

The Locus of Emotional-Processing Abnormalities in PTSD

Contemporary models of emotion posit that affective reports and behavior are organized at a fundamental level by the activation of appetitive and defensive motivational systems (Cacioppo, Gardner, & Berntson, 1999; Davidson, Jackson, & Kalin, 2000; Lang, Bradley, & Cuthbert, 1997). Lang and colleagues have labeled this basic level of emotional activation *strategic* (Lang et al., 1990). Within this framework, specific affects such as fear or anger are viewed as subordinate organizations of the overarching strategic disposition that becomes differentiated at a tactical level through expressive behaviors that vary by the demands of specific contexts and the learning history of the individual.

Theorists have speculated that the numbing symptoms of PTSD may reflect a form of emotional avoidance that serves to reduce the likelihood of experiencing intense affect (e.g., Barlow, 2002; Hayes, Wilson, Gifford, Follette, & Strosahl, 1996). Support for this notion was provided by evidence that anxious individuals tend to avoid arousing emotions (Williams, Chambless, & Ahrens, 1997), which suggests both that individuals with PTSD may deliberately conceal or withhold the expression of emotion (Roemer, Litz, Orsillo, & Wagner, 2001) and that the tactical expression of emotion can be intentionally modulated in humans (Bradley, Codispoti, Cuthbert, & Lang, 2001). Thus, one might hypothesize that reduced emotional expressivity following exposure to trauma-related cues reflects an affective abnormality operating at Lang et al.'s (1990) tactical, as opposed to strategic, level of emotional response.

Alternatively, emotional-processing abnormalities in PTSD could reflect alterations in activation of the basic (strategic) appetitive and aversive motive systems. Animal research on the motivational impact of conditioned aversive stimuli has shown that presentation of a CS+ produces phasic decrements in appetitive motivation, a phenomenon known as *conditioned suppression* (Estes, 1969; Estes & Skinner, 1941; Millenson & de Villiers, 1972). By extension, in PTSD, activation of a trauma-related CER might be expected to inhibit subsequent appetitive responses at the fundamental strategic level at which reflexes are primed. During picture processing, this would be reflected in less startle attenuation during viewing of pleasant pictures after exposure to a trauma-related stressor relative to during a baseline interval.

On the other hand, there is also evidence that patterns of startle modulation are sensitive to manipulations of negative affect and that when the defensive system is primed the response to unpleasant emotional stimuli may be synergistically intensified in anxious individuals (Miller & Patrick, 2000). If this is the case, we might also expect to observe enhanced startle potentiation to unpleasant stimuli after exposure to a trauma-related challenge in individuals with PTSD.

Primary Aims and Hypotheses of the Study

The primary objective of this study was to conduct a psychophysiological assessment of emotional behavior in PTSD to replicate and extend our previous work and to clarify the locus of emotional-processing abnormalities in PTSD. Following Litz et al. (2000), we predicted state-dependent, emotional-processing abnormalities to be characteristic of individuals with PTSD. Specifically, individuals with PTSD were expected to exhibit suppressed emotional responses to pleasant stimuli and augmented responses to unpleasant stimuli compared with control individuals after exposure to a trauma-related challenge. Moreover, we hypothesized that if the impact of the CER operates primarily at the tactical level of emotion-influencing expressive behavior, then we should expect to observe group differences in patterns of facial EMG (e.g., suppressed zygomatic and enhanced corrugator EMG activity during processing of pleasant and unpleasant images, respectively) but not in patterns of affect-modulated startle. On the other hand, if the impact of the CER operates at the strategic level at which reflexes are primed (i.e., via conditioned suppression) then we should expect to also observe alterations in patterns of reflex modulation (i.e., less startle response inhibition and greater potentiation during viewing of pleasant and unpleasant pictures, respectively).

Trauma-Context Specificity of the Hypothesized Effects

The second aim of this study was to examine whether hypothesized changes in emotional behavior are specific to the trauma-related content of the experimental manipulation or a nonspecific consequence of heightened negative affectivity. Litz et al. (2000) compared emotional responses under neutral conditions with those that followed a trauma-related challenge but were unable to evaluate whether the influence of a trauma challenge on subsequent emotional behavior was specific to the content of the manipulation or merely a consequence of heightened negative affect. To address this issue, we used a non-trauma-related stressor (i.e., threat of electric shock) in addition to the trauma-related challenge.

Objectives of the Stress Manipulations

Our objectives for this aspect of the design were to implement a non-trauma-related stressor that would elicit comparable levels of physiologic reactivity in participants with and without PTSD and to develop trauma-related and non-trauma-related challenges that would evoke comparable levels of negative affective arousal in the non-PTSD control group. (Given that physiologic hyperreactivity to trauma-related cues is syndromal in PTSD, we did not expect the two groups to exhibit equivalent responses to the trauma-related challenge.) We chose threat of shock as the non-trauma-related stressor because it was expected to conjure few, if any, associations to combat-related trauma and because prior studies have shown it to be a potent manipulation of negative affective arousal (e.g., Ameli et al., 2001; Grillon, Ameli, Merikangas, Woods, & Davis, 1993; Miller & Patrick, 2000). On the basis of the hypothesis that alterations in patterns of emotional response in individuals with PTSD are specific to, and a consequence of, activation of a trauma-related CER (Litz, 1992; Litz et al., 2000) we predicted that changes in positive/appetitive and negative/defensive emotional reactivity would be evident only after exposure to the trauma-related challenge.

Method

Participants

Participants were 59 male combat-exposed veterans of either the Vietnam War ($n = 47$) or the Gulf War ($n = 12$) recruited through flyers posted at a Veterans Affairs Medical Center. Participants were assigned to a current combat-related PTSD group ($n = 35$; 7 Gulf War veterans) or a non-PTSD group ($n = 24$; 5 Gulf War veterans) on the basis of symptoms specified in the *DSM-IV* (American Psychiatric Association, 1994), endorsed with an intensity of 1 or greater, and a frequency of 2 or greater within the last month on the *Clinician Administered PTSD Scale* (CAPS; frequency and intensity values range from 0–4; Weathers, Keane, & Davidson, 2001). The two groups were matched on age and lifetime PTSD was not assessed. Individuals were excluded if they could not hear 125–8000 Hz tones presented over headphones at 70 dB or less or if they reported psychotic symptoms on the psychotic screen of the *Structured Clinical Interview for DSM–IV* (SCID; First, Spitzer, Gibbon, & Williams, 1996). Two individuals were excluded because they failed the hearing screen; none were excluded as a result of psychosis.

Individuals were also excluded from participation if they reported during a prescreening interview that they were taking beta-blockers (e.g., propranolol or atenolol). The presence of benzodiazepines was assessed with a urine toxicology screen administered during the laboratory session; 4 participants in the PTSD group and 2 participants in the non-PTSD group tested positive for this compound. Other medication status was assessed using a self-report checklist. Endorsement rates for other major classes of psychiatric medication were as follows: other anxiolytics (2 PTSD; 0 non-PTSD); SSRIs (9 PTSD; 1 non-PTSD), other antidepressants (11 PTSD; 2 non-PTSD); anticonvulsants (2 PTSD; 0 non-PTSD).

Experimental Design

The experiment used a mixed factorial design consisting of a two-level between-subjects factor (group: PTSD, non-PTSD), and two three-level within-subjects factors, (block: baseline, post-shock stressor, post-trauma-related stressor) and (picture valence: pleasant, neutral, and unpleasant). Each block of the procedure contained 18 pictures with 6 in each valence category. Picture valence was balanced at each serial position across three counterbalancing orders. The non-trauma-related stressor occurred between Blocks 1 and 2; the trauma-related stressor took place between Blocks 2 and 3. The order in which participants underwent the trauma-related and non-trauma related challenges was fixed.[1]

Procedure

The study was conducted over two sessions scheduled approximately 1 week apart. The first was devoted to diagnostic interviews and questionnaire administration, the second to the laboratory assessment. When participants arrived for the first session, they were informed about the nature

[1] The primary reason for presenting the challenges in a fixed order (rather than alternating the order between subjects) was to avoid introducing powerful between-subject counterbalancing order effects related to the possibility that the two challenges would have differential carryover effects on subsequent phases of the procedure. In addition, the fixed order was designed to permit comparison of the patterns of results for blocks one and two in this study with our previous one (Litz et al., 2000) in which the procedures were virtually identical with the exception of the nature of the stressor.

and requirements of the study and completed an Institutional Review Board (IRB)-approved consent form. They were then interviewed using the CAPS and SCID psychotic screen and major depression modules. Interviewers were postdoctoral-level clinical psychologists with extensive experience with the administration of both instruments. Participants then completed a series of self-report instruments designed to measure war-zone exposure (Combat Exposure Scale; Keane et al., 1989), PTSD (PTSD Checklist; Weathers, Litz, Huska, & Keane, 1991) and co-morbid symptomatology (Beck Anxiety and Depression Inventories; T. Beck, Epstein, Brown, & Steer, 1988; A. T. Beck, Ward, Mendelsohn, Mock, & Erbaugh, 1961), mood state (Positive and Negative Affect Schedule; PANAS, present state version; Watson, Clark, & Tellegen, 1988), personality characteristics (Multidimensional Personality Questionnaire—Brief Form; MPQ; Patrick, Curtin, & Tellegen, 2002), alcohol-related problems (Short Michigan Alcoholism Screening Test; SMAST; Selzer, 1971; Zung, 1979) and substance abuse within the previous 12 months (Drug Abuse Screening Test; DAST; Skinner, 1982).

The psychophysiological assessment took place in a dimly lit, sound attenuated chamber with participants seated upright in a comfortable chair located 3 ft (0.9 m) in front of a 20-in. (50.8 cm) computer monitor. A computer mouse and pad was attached to the arm of the chair. Figure 1 illustrates the design of the laboratory procedure. The session began with demonstration of the Self-Assessment Manikin (Lang, 1980) rating procedure, administration of a baseline PANAS, electrode attachment, and headphone placement. Participants then viewed the first block of pictures (6 pleasant, 6 neutral, and 6 unpleasant) while their physiological responses were recorded with the instruction to "view each picture for the entire time that it is on the screen and then rate how you felt while looking at it." Participants received no specific instructions about the location or nature of the shock and trauma-related stress periods prior to the first block. Instructions for the stress periods were delivered immediately prior to the commencement of those periods.

Each photographic image was displayed for 6 s, followed by a 6-s blank screen, then presentation of the Self-Assessment Mannikin (SAM) valence followed by arousal ratings. One startle probe was presented during each picture at either 3.5, 4.5, or 5.5 s after image onset (counterbalanced with valence) for a total of 18 probes per block with 6 images in each valence category. Participants were instructed to try to ignore the startle probes. During each block, nine additional startle probes were presented during intervals in which no picture was present (ITIs). Three of these probes occurred during a 90-s interval prior to the first picture. The remaining probes were interspersed at unpredictable intervals during the block with serial position held constant across counterbalancing orders.

After completing the first (baseline) block, shock electrodes were attached to the second and fourth fingers of the participant's nondominant hand for the non-trauma-related stressor with the instruction that "during the next 5-min period, at least one, but not more than three, moderately painful but not harmful electric shocks will be delivered without warning." In fact, only one shock was actually delivered, occurring 30 s into the shock-threat interval. The purpose of administering the shock was to equate participants' expectations about its intensity and to enhance the validity of the threat manipulation. The 5-min shock period was signaled by the word *shock* displayed on the monitor throughout the interval. Physiological data were collected throughout the 5-min shock-threat interval and during a 90-s prestress baseline interval prior to display of the word *shock*. Three startle probes were presented during the prestress baseline interval at unpredictable intervals averaging 30-s in duration. Five more were delivered during the 5-min threat period at intervals averaging 1 min in duration.

When the shock stressor ended the experimenter removed the shock electrodes, administered a second PANAS, and initiated the second block of pictures during which physiological and self-report responses were recorded as before. The second block was followed by the 5-min trauma-related stressor that consisted of a series of combat-related photographs presented concurrently with combat-related audio recordings. As before, physiological data were recorded during a 90-s baseline interval prior to the onset of the audiovisual presentation, and throughout the 5-min challenge. Three startle probes were presented during the baseline interval and five probes were presented during the audiovisual presentation. The latter were delivered during 2-s intervals when no picture or sounds were present. When this was over, a third PANAS was administered, followed by a third and final block of pictures with physiological recordings. After the third block, participants completed a fourth PANAS and a postexperiment questionnaire assessing reactions to the experimental stimuli. They were then debriefed and paid for their time.

Stimulus Materials

Photographic images. Digitized images were selected from the International Affective Picture System (IAPS; Center for the Study of Emotion and Attention [CSEA–NIMH], 1999) on the basis of normative ratings for males (Lang, Bradley, & Cuthbert, 1999). Although different sets of pictures were used in each block, pleasant and unpleasant images were matched for mean (high) arousal both within and across blocks. Pleasant and unpleasant images had mean valence ratings that were equidistant from neutral.

The pleasant category was comprised of two types of images: erotic images depicting heterosexual couples engaged in sexual behaviors or female nudes, and nurturant images depicting cute babies or pets. Erotic images were chosen on the basis of prior work showing that they were maximally effective for producing inhibition of the startle response (e.g., Bradley et al., 2001). Nurturant images were selected because they evoke strong zygomatic EMG responses and relate to pleasant interpersonal experiences that are not tapped by the erotic stimuli and are defined by the *DSM–IV* as central to the emotional deficits of patients with PTSD. Four erotic and two nurturant photos were presented in each block.

The unpleasant category was comprised of images depicting the following: (a) mutilated human bodies, (b) threats to self (involving threats directed toward the viewer including attacking animals, aimed guns, and looming assailants), and (c) assaults on others (scenes of people being attacked violently). These subcontents were distributed equally in each

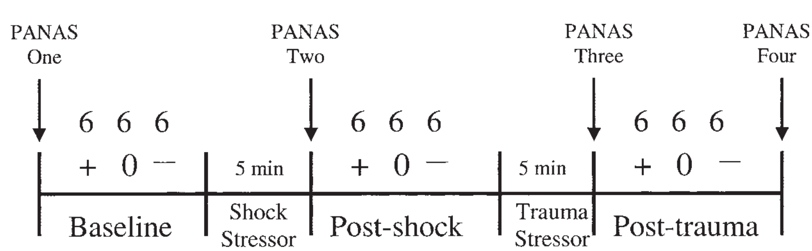

Figure 1. Schematic diagram of the procedure. PANAS = Positive and Negative Affect Schedule.

block. Neutral images included household objects and other familiar stimuli.[2]

Trauma-related stressor. Two 5-min multimedia presentations depicting scenes of combat were created with scenes of the Vietnam War and the Gulf War, respectively. Both consisted of 30 photographic images depicting traumatic scenes of war and its aftermath, including military personnel and/or civilians who were in distress, wounded, or dead, presented continuously for 10 s each. The images were displayed concurrently with 5 min of war-related sounds that were digitally recorded from audio tracks of war documentaries and normalized for uniformity of sound level.

Noise and shock stimuli. The acoustic startle probe consisted of a 50-ms burst of 104 dB white noise with immediate (< 10 ms) rise time. The stimulus was produced by a Coulbourn Instruments (Allentown, PA) white noise generator (Model S81–02), amplified by an audio mixer-amplifier (Model S82–24), and presented binaurally through headphones. Electric shocks were generated by a Coulbourn Instruments transcutaneous aversive finger stimulator (Model E13–22). The stimulus was delivered at an intensity of 5.0 mA, with a frequency of 10 pulses per second, for a duration of 500 ms via electrodes attached to the second and fourth fingers of the participant's nondominant hand.

Apparatus, Recording, and Data Reduction

The SuperLab software program (Cedrus Corporation, 1999) using a 1000-Hz timer was used to display digital images, collect ratings data, coordinate the onset and offset of data collection, and control the timing of all experimental stimuli. The Labtech Notebook Pro software program (Labtech Corporation, 2000) running on a second computer controlled the sampling, digitization, and storage of physiological data. The onset and offset of physiological data collection was controlled by a transistor-transistor logic (TTL) signal from the computer running the Superlab program.

Startle response. The eyeblink component of the startle reflex was measured by recording EMG activity from Beckman miniature Ag/AgCl electrodes positioned over the orbicularis oculi muscle beneath the left eye. The raw EMG signal was amplified with a bioamplifier (Model S75–01) with low- and high-frequency cutoffs of 90 and 1000 Hz, respectively. The signal was rectified and integrated with a contour following integrator (Model S76–01) with a time constant setting of 100 ms. Digital sampling commenced at 1000 Hz 50 ms before startle probe onset and continued for 150 ms after probe offset. The startle response data were reduced off-line using a program developed by Curtin (1996) which scores startle-elicited blinks for magnitude in arbitrary analog–digital (A–D) units.

To control for individual differences in overall blink magnitude and rate of habituation across the three blocks of the experiment, startle responses recorded during the picture processing periods were standardized within each block using a within-participant *z*-score transformation. Startle responses recorded during the stress manipulations were untransformed. Analyses of these data focused on the difference between the mean blink magnitude for three probes presented during the 90-s baseline intervals prior to the onset of the stressor compared with the mean response to five probes presented during the 5-min stress manipulations. Startle response data were missing for 3 participants as a result of equipment malfunction. Data for 3 additional participants (2 non-PTSD) were eliminated because more than 50% of trials within a block of 27 were scored as no response or rejected because of movement artifact.

Heart rate. HR activity was recorded from 1-cm Beckman Ag/AgCl electrodes positioned on the right and left inner forearms. The signal was filtered with a Coulbourn bioamplifier (Model S75–01) with high and low filter settings of 8 and 40 Hz, respectively. A Schmitt trigger interrupted the computer each time it detected the R component of the cardiac waveform. Interbeat intervals were recorded in milliseconds and reduced online to HR in beats per minute (BPM) with 0.5-s intervals. HR data were screened for artifact/outliers using a two-step process. In the first pass, data values that fell out of the range of 45–100 BPM and/or consecutive sampling values that differed by 24 BPM or more were set to missing (Berntson, Quigley, Jan, & Boysen, 1990). In the second pass, missing observations were replaced with a linear interpolation function based on surrounding valid data points over segments of up to 6 consecutive observations (i.e., 3 s). Change scores for the picture processing procedure were computed by subtracting the mean of the 6-s baseline interval from the mean of the 6-s picture-viewing interval. For the trauma-related and non-trauma-related stress periods, analyses examined the difference between the mean of the 30-s baseline interval immediately prior to stressor onset and the mean of the maximum 10-s interval during the 5-min stress period. HR data were missing for 3 participants as a result of equipment malfunction.

Skin conductance. SC was recorded from adjacent sites on the hypothenar eminence of the non-dominant hand with 1-cm Beckman Ag/AgCl electrodes filled with Unibase–saline paste (Lykken & Venables, 1971) and connected to a Coulbourn isolated SC coupler (Model S71–23). During the picture-viewing procedure, digital sampling (10 Hz) commenced 1-s prior to the onset of the image and continued through the 6-s presentation interval. Change in SC level (SCL) during picture processing was computed by subtracting the mean of the 1-s baseline interval prior to the onset of the picture from the mean of the 6-s picture-viewing interval. As with HR, change in SCL in response to the two stressors was computed by subtracting the mean of the 30-s baseline from the mean of the maximum 10-s interval during the 5-min stress period.

Facial EMG. Corrugator and zygomatic EMG activity was recorded from Beckman miniature Ag/AgCl electrodes filled with electrolyte paste positioned according to standard guidelines (Fridlund & Cacioppo, 1986). The raw EMG signals were amplified with a bioamplifier (Model S75–01) with low- and high-frequency settings of 90 and 1000 Hz, respectively, and then rectified and integrated (time constant = 500 ms). During the picture-viewing procedure, EMG activity was sampled at 10 Hz beginning 1 s prior to picture onset, and it continued through the 6-s viewing period. Facial EMG change scores were computed by subtracting the mean of the 1-s baseline interval prior to picture onset from the mean of the 6-s picture-viewing interval. Change in corrugator activity in response to the two stressors was computed in the same manner as was described for HR and SC.

Data Analysis

Primary hypotheses for each dependent measure during picture processing were tested using a multivariate analysis of variance (MANOVA) with repeated measures treated as variates (Stevens, 1992; Vasey & Thayer, 1987), and effects were assessed for significance using the Wilks's lambda statistic. For all measures other than startle, the picture valence factor was parsed into orthogonal "quadratic" (i.e., pleasant/unpleasant vs. neutral) and "linear" (i.e., pleasant vs. unpleasant) contrasts—reflecting effects of arousal/activation and emotional valence, respectively (cf. Bradley et al., 1990; Miller et al., 2002; Miller & Patrick, 2000). For startle analyses, the neutral valence category was replaced with data for ITI (i.e., no picture) probes. In this case, the quadratic contrast (i.e., pleasant/unpleasant vs. ITI) provided (a) an index of the degree to which startle responses were suppressed during picture processing and (b) a measure of the degree of engagement in processing of the visual foreground stimulus.

[2] IAPS images used were as follows: Pleasant (nurturant): 1463, 1710, 1750, 2050, 2057, 2070. Pleasant (erotic): 4001, 4002, 4235, 4290, 4300, 4302, 4310, 4651, 4658, 4659, 4670, 4800. Neutral: 2200, 5500, 5510, 6150, 7002, 7010, 7050, 7060, 7080, 7090, 7100, 7130, 7150, 7170, 7490, 7700, 7710, 9070. Unpleasant (mutilations): 3000, 3010, 3053, 3060, 3080, 3170. Unpleasant (threat to self): 1050, 1300, 6243, 6250, 6370. Unpleasant (assaults on others): 2681, 3500, 3530, 6313, 6350, 6560, 6821.

Results

Participant Characteristics

Table 1 lists descriptive information for the two study groups. No significant group differences were found for age or race/ethnicity. The PTSD group scored higher than the non-PTSD group on all measures of combat exposure and psychopathology including PTSD, major depression, anxiety, and alcohol and substance abuse. On the MPQ, participants in the PTSD group produced significantly higher scores on Negative Emotionality and lower scores on Positive Emotionality and Constraint compared with those of non-PTSD participants.

Stress Manipulations

Self-report responses. Table 2 lists the mean PANAS scores over the course of the procedure by group. The PANAS was administered at four points during the procedure: (a) at baseline, (b) after the non-trauma-related (shock) stressor, (c) after the trauma-related stressor, and (d) at the end of the procedure. Positive affect (PA) and negative affect (NA) scores were analyzed separately with repeated measures ANOVAs using a four-level within-subject factor reflecting the four assessment points (Time), and the two-level between-subject factor PTSD group. For PA, analyses revealed a significant effect of time, $F(3, 53) = 7.04$, $p < .01$, indicating that scores tended to drop over the course of the procedure. This effect did not differ as a function of group. For NA, analyses revealed a main effect of time, $F(3, 53) = 20.2$, $p < .01$, and a significant Time × Group interaction, $F(3, 53) = 3.30$, $p < .03$. Decomposition of the interaction revealed that it was accounted for primarily by a larger NA increase from postshock to posttrauma-stressor periods in the PTSD group. That is, while the Time × Group interaction for the baseline to postshock assessment was nonsignificant, $F(1, 57) = 1.0$, this interaction was significant for the postshock to posttrauma comparison, $F(1, 57) = 6.91$, $p < .02$. The PTSD group showed a larger increase in NA, $F(1, 34) = 46.05$, $p < .001$, across the two intervals than the non-PTSD group, $F(1, 23) = 8.81$, $p < .01$.

Table 2
Mean PANAS Scores (and Standard Deviations) Over the Course of the Procedure, by Group

	Positive affect		Negative affect	
Assessment	Non-PTSD (n = 24)	PTSD (n = 35)	Non-PTSD (n = 24)	PTSD (n = 35)
Baseline	31.9 (6.6)	31.8 (8.6)	11.3 (1.7)	15.2 (5.0)
Postshock	30.8 (9.2)	32.2 (9.0)	12.3 (3.3)	18.0 (7.6)
Posttrauma	30.6 (9.0)	31.8 (9.3)	16.7 (6.5)	28.1 (10.4)
End	27.7 (9.4)	30.0 (9.8)	12.3 (3.0)	19.9 (9.1)

Note. PANAS = Positive and Negative Affect Schedule; PTSD = posttraumatic stress disorder.

Physiological responses. Tables 3 and 4 list the mean response to the trauma-related and non-trauma-related stressors for each physiological measure by group. Three-way repeated measures analyses including the two-level within-subject factor, stress (i.e., prestressor baseline vs. stress period), and condition (non-trauma-related [shock] vs. trauma-related stressor), and between-subject factor PTSD group were used to test the immediate impact of the stress manipulations (as opposed to the subsequent effect on picture processing, which is reported below). For corrugator EMG, analyses revealed a main effect of stress that was unmodified by condition or group, $F(1, 56) = 31.22$, $p < .001$. For HR, there was a main effect of stress, $F(1, 54) = 86.70$, $p < .01$, and a trend toward a Condition × Stress × PTSD interaction, $F(1, 54) = 3.46$, $p < .07$. A test of the a priori hypothesis that participants with PTSD would exhibit greater HR increases during the trauma-related stressor was supported by a significant Stress × PTSD interaction, $F(1, 54) = 4.15$, $p < .05$. No such interaction was evident in the data for the shock stressor. For SC, there was a main effect of stress, $F(1, 54) = 67.60$, $p < .001$, and a significant Condition × Stress interaction, $F(1, 54) = 14.27$, $p < .001$, indicating that SC increases were larger during the shock stressor than during the trauma-related manipulation. Finally, for startle, the mean amplitude of the response to three probes presented during each 90-s prestress baseline interval was compared with the mean response to the five probes presented during each 5-min stress period. These analyses revealed a main effect of condition, $F(1, 51) = 69.19$, $p < .001$, with larger startle responses observed during the shock versus trauma manipulation, and a significant Condition × Stress interaction, $F(1, 51) = 50.59$, $p < .001$, indicating that startle responses were modulated in opposite directions during the two stressors (i.e., potentiated during the shock

Table 1
Means (and Standard Deviations) of Descriptive Characteristics for the Two Study Groups

Variable	Non-PTSD (n = 24)	PTSD (n = 35)	t
Demographic measures			
Age	52.60 (8.20)	51.00 (7.30)	*ns*
Combat Exposure Scale	11.75 (6.25)	15.66 (5.67)	2.50*
Race/ethnicity (%)			
Caucasian	79.00	67.00	*ns*
African American	17.00	26.00	*ns*
Hispanic/other	4.00	7.00	*ns*
PTSD measures			
CAPS total	19.46 (15.30)	73.80 (19.49)	11.44**
PTSD Checklist	31.08 (14.19)	58.24 (12.30)	7.71**
Other psychopathology measures			
Alcohol abuse (SMAST)	2.58 (3.99)	5.23 (4.17)	2.44*
Beck Anxiety Inventory	6.87 (8.49)	21.74 (12.55)	5.05**
Beck Depression Inventory	6.08 (6.27)	23.94 (11.39)	6.98**
Drug abuse (DAST)	0.71 (1.20)	2.06 (2.88)	2.16*
SCID major depression (%)	4.50	54.50	3.79**
MPQ			
PEM	52.96 (11.33)	35.94 (10.47)	5.89**
NEM	49.38 (8.27)	65.91 (7.15)	8.13**
CON	48.71 (9.27)	42.15 (8.54)	2.78*

Note. PTSD = posttraumatic stress disorder; CAPS = Clinician Administered PTSD Scale; SMAST = Short Michigan Alcoholism Screening Test; DAST = Drug Abuse Screening Test; SCID = Structured Clinical Interview for Axis I and Axis II *DSM–IV* Disorders; MPQ = Multidimensional Personality Questionnaire; PEM = Positive Emotionality; NEM = Negative Emotionality; CON = Constraint. The dichotomous SCID major depression variable was analyzed using the nonparametric Mann–Whitney *U* test.
* $p < .05$. ** $p < .01$.

Table 3
Mean Physiological Responses (and Standard Errors) During the Non-Trauma-Related Stressor (Shock), by Period (Prestress vs. Stress Period) and Group

	Non-PTSD (n = 22)				PTSD (n = 34)			
	Prestress baseline		Stress period		Prestress baseline		Stress period	
Measure	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*
Startle amplitude (μV)	12.83	1.72	14.62	1.82	12.38	1.86	14.14	1.79
HR (BPM)	74.83	2.78	80.06	3.14	73.35	1.94	78.89	1.99
SCL (μS)	0.50	0.08	0.62	0.09	0.45	0.06	0.55	0.07
CORR (μV)	0.22	0.02	0.60	0.12	0.21	0.02	0.50	0.06

Note. PTSD = posttraumatic stress disorder; HR = heart rate; BPM = beats per minute; SCL = skin conductance level; CORR = corrugator electromyography response.

stressor and inhibited during the trauma-related stressor). This effect was not modified by group.

Picture-Viewing Procedure

Startle reflex modification. Table 5 lists means for all dependent measures by picture valence (pleasant, neutral/ITI, unpleasant) and block (baseline, postshock, posttrauma). A three-way (PTSD Group × Trial Block × Valence) repeated measures MANOVA performed on the standardized blink magnitude scores revealed a main effect of valence, $F(2, 56) = 27.29$, $p < .01$, with startle responses reliably potentiated during unpleasant compared with pleasant pictures, linear $F(1, 57) = 17.67$, $p < .01$, and inhibited during picture viewing relative to ITI, quadratic $F(1, 57) = 40.43$, $p < .01$, and a significant Group × Block × Valence interaction, $F(4, 54) = 3.24$, $p < .02$.

When the three-way interaction was decomposed by examining the patterns of means within each block separately, data from the baseline period revealed a main effect of valence, $F(2, 56) = 28.12$, $p < .01$, with significant linear and quadratic trends (Fs = 13.72 and 56.83, respectively) but these effects were unmodified by group. A similar valence effect, also unmodified by group, was observed during the postshock period, $F(2, 56) = 19.16$, $p < .01$ (linear and quadratic Fs = 13.78 and 18.92, respectively). Following the trauma-related stressor, however, analyses revealed a significant Valence × PTSD interaction, $F(2, 56) = 3.96$, $p < .03$. Examination of the pattern of means in this block for each group separately showed that the non-PTSD group exhibited a significant quadratic valence effect with startle responses attenuated during pictures relative to probes presented during ITIs, $F(1, 23) = 8.91$, $p < .007$, with no significant difference between pleasant and unpleasant. In contrast, the PTSD group exhibited a significant linear effect, $F(1, 34) = 4.32$, $p < .05$, indicating that blink responses were significantly larger during processing of unpleasant than pleasant images with no quadratic trend. In other words, in the PTSD group, during Block 3, ITI startles were intermediate in amplitude compared with during viewing of pleasant and unpleasant images.

To further characterize these effects and to examine the degree to which engagement in processing of the foreground picture stimulus varied across blocks, we conducted a second set of analyses that examined picture – ITI difference scores for pleasant and unpleasant pictures separately. This pattern of means is depicted in Figure 2. Analysis of the pleasant – ITI difference scores revealed a significant block effect, $F(2, 56) = 5.53$, $p < .01$, indicating that the degree of blink attenuation during viewing of pleasant images tended to dissipate over the course of the procedure, but there were no main or interactive effects of group. In contrast, analysis of the unpleasant – ITI difference scores revealed

Table 4
Mean Physiological Responses (and Standard Errors) During the Trauma-Related Stressor, by Period (Prestress vs. Stress Period) and Group

	Non-PTSD (n = 23)				PTSD (n = 34)			
	Prestress baseline		Stress period		Prestress baseline		Stress period	
Measure	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*
Startle amplitude (μV)	6.12	0.81	2.09	0.54	6.77	1.39	2.78	0.54
HR (BPM)	74.81	2.92	78.18	2.78	71.78	1.98	78.70	1.90
SCL (μS)	0.48	0.07	0.53	0.08	0.42	0.06	0.48	0.07
CORR (μV)	0.16	0.02	0.64	0.12	0.18	0.02	0.64	0.12

Note. PTSD = posttraumatic stress disorder; HR = heart rate; BPM = beats per minute; SCL = skin conductance level; CORR = corrugator electromyography response.

Table 5
Mean Physiological and Self-Report Responses (and Standard Errors) During Picture Viewing Across Measures, by Block, Valence, and Group

	Block 1 (baseline)			Block 2 (postshock)			Block 3 (postprime)		
Measure and group	P	ITI or N	U	P	ITI or N	U	P	ITI or N	U
Physiological measures									
Startle amplitude (*z* scores)									
Non-PTSD (n = 24)									
M	−0.28	0.30	0.06	−0.24	0.31	−0.09	−0.20	0.21	−0.16
SE	0.07	0.05	0.06	0.05	0.08	0.09	0.08	0.08	0.07
PTSD (n = 35)									
M	−0.35	0.28	−0.09	−0.33	0.15	0.11	−0.14	−0.02	0.11
SE	0.07	0.05	0.06	0.05	0.07	0.08	0.07	0.06	0.08
HR change (BPM)									
Non-PTSD (n = 23)									
M	−0.51	−0.49	−0.57	−0.40	−0.48	−1.21	−0.49	−0.14	−1.27
SE	0.28	0.23	0.24	0.36	0.35	0.22	0.33	0.30	0.36
PTSD (n = 33)									
M	0.10	−0.10	−0.04	−0.46	−0.03	−0.29	0.06	−0.70	−0.68
SE	0.18	0.17	0.25	0.31	0.32	0.29	0.20	0.27	0.31
SC change (μS)									
Non-PTSD (n = 24)									
M	0.019	0.004	0.016	0.008	0.004	0.012	0.007	0.004	0.002
SE	0.005	0.003	0.004	0.002	0.002	0.004	0.004	0.003	0.002
PTSD (n = 35)									
M	0.007	0.002	0.011	0.006	−0.001	0.004	0.002	0.001	0.004
SE	0.002	0.001	0.002	0.002	0.001	0.001	0.001	0.001	0.001
Zygomatic change (μV)									
Non-PTSD (n = 24)									
M	0.44	0.08	0.06	0.26	0.12	0.12	0.22	0.16	0.02
SE	0.14	0.06	0.08	0.10	0.08	0.08	0.10	0.10	0.12
PTSD (n = 34)									
M	0.34	0.10	0.10	0.26	0.00	0.02	0.30	−0.02	0.22
SE	0.12	0.10	0.08	0.16	0.04	0.08	0.08	0.12	0.12
Corrugator change (μV)									
Non-PTSD (n = 24)									
M	0.04	0.24	0.38	−0.04	0.22	0.42	−0.14	−0.34	0.24
SE	0.18	0.08	0.16	0.28	0.14	0.20	0.18	0.38	0.22
PTSD (n = 34)									
M	0.08	0.24	0.50	0.04	0.48	0.72	0.22	0.78	0.84
SE	0.12	0.10	0.20	0.20	0.18	0.28	0.14	0.32	0.36
Ratings									
Valence or pleasantness									
Non-PTSD (n = 23)									
M	7.45	5.14	2.62	7.23	5.14	2.92	7.27	4.80	2.73
SE	0.21	0.23	0.21	0.16	0.13	0.21	0.23	0.14	0.20
PTSD (n = 34)									
M	7.07	5.23	2.67	6.97	5.04	2.90	6.63	4.75	3.05
SE	0.21	0.11	0.24	0.23	0.12	0.24	0.25	0.16	0.29
Arousal									
Non-PTSD (n = 23)									
M	4.69	2.14	5.30	4.73	2.25	5.23	4.89	2.67	5.32
SE	0.40	0.24	0.40	0.43	0.23	0.42	0.44	0.20	0.42
PTSD (n = 34)									
M	4.69	2.56	5.80	4.72	2.46	5.53	4.31	2.88	5.52
SE	0.36	0.27	0.38	0.38	0.27	0.37	0.35	0.29	0.39

Note. For startle response, means reported in the middle column are for intertrial interval (ITI) probes. The means and standard errors of the startle responses to neutral slides in standardized scores were the following (by block [B]): Non-PTSD, (B1: −0.29 [0.07]; B2: −0.13 [0.09]; B3: 0.02 [0.09]); PTSD (B1: 0.06 [0.07]; B2: −0.01 [0.07]; B3: 0.03 [0.07]). *Z* scores for startle blink amplitude do not necessarily sum to zero because data for the neutral condition were included in the transformation but were omitted from the table. Ratings data are based on 9-point Self-Assessment Manikin scales (Lang, 1980). P = pleasant; N = neutral; U = unpleasant; PTSD = posttraumatic stress disorder; HR = heart rate; BPM = beats per minute; SC = skin conductance.

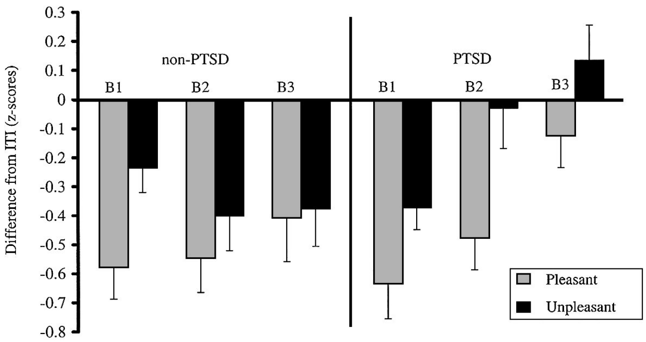

Figure 2. Startle responses to acoustic probes presented during viewing of pleasant and unpleasant pictures relative to responses on no-picture trials by block and group. ITI = intertrial interval; PTSD = posttraumatic stress disorder; B = block.

a main effect of group, $F(1, 57) = 4.26$, $p < .05$, indicating less attenuation of the startle response (i.e., relatively larger startles) in the PTSD group during viewing of unpleasant pictures. This effect was modified by an interaction with block, $F(2, 56) = 6.43$, $p < .01$, that was decomposed by comparing group means for each block separately through the use of *t* tests with equal variances assumed. Results showed no significant group differences in the unpleasant – ITI difference during the baseline or postshock periods. During the posttrauma challenge period, however, analyses showed that individuals with PTSD exhibited significantly larger relative blink amplitudes (i.e., a smaller unpleasant – ITI difference) during viewing of unpleasant images compared with those exhibited by the non-PTSD group, $t(57) = 2.87$, $p < .01$.

HR response. A three-way (PTSD Group × Trial Block × Valence) repeated measures MANOVA performed on the 6-s HR change scores revealed a main effect of valence, $F(2, 52) = 3.61$, $p < .04$, with greater HR deceleration observed during viewing of unpleasant relative to pleasant images: linear valence, $F(1, 59) = 5.92$, $p < .02$. This effect was modified by block indicating that the magnitude of the difference between pleasant and unpleasant increased over the course of the procedure, $F(1, 59) = 5.56$, $p < .02$. Results also showed a main effect of group, $F(1, 53) = 4.49$, $p < .04$, with greater HR deceleration observed in the non-PTSD group irrespective of valence relative to the PTSD group.

SCL. Analyses performed on the range-corrected 6-s SCL change scores revealed several effects of interests. First, there was a significant main effect of valence, $F(2, 56) = 20.63$, $p < .001$, with larger SC increases during viewing of pleasant and unpleasant pictures relative to neutral, quadratic valence, $F(1, 57) = 40.90$, $p < .001$ and no overall difference between pleasant and unpleasant categories. Analyses also revealed a significant main effect of block, $F(2, 56) = 10.72$, $p < .01$, and a significant Block × Valence interaction, $F(4, 54) = 6.65$, $p < .001$, reflecting a tendency for SCL increases to diminish over the course of the procedure. There was also a significant Block × Valence × PTSD interaction, $F(4, 54) = 3.51$, $p < .02$; however, decomposition of this effect revealed no reliable group differences during any individual block.

Zygomatic EMG response. Analysis of the zygomatic EMG change scores revealed a main effect of valence, $F(2, 55) = 4.18$, $p < .02$, with significant linear, $F(1, 56) = 5.70$, $p < .03$, and quadratic trends, $F(1, 56) = 6.17$, $p < .02$, indicating that pleasant images were associated with greater zygomatic increases than unpleasant ones and that zygomatic increases were greater during viewing of pleasant and unpleasant stimuli relative to neutral. There were no significant block or group effects.

Corrugator EMG response. Analysis of the corrugator EMG change scores revealed a main effect of valence, $F(2, 55) = 4.13$, $p < .02$, with a significant linear trend, $F(1, 56) = 8.41$, $p < .006$, indicating that unpleasant slides were associated with greater corrugator increases than were pleasant slides. There was also a significant Block × PTSD interaction, $F(2, 55) = 3.87$, $p < .03$. Decomposition of this interaction revealed that participants in the PTSD group exhibited significantly larger corrugator responses irrespective of valence content during the posttrauma block relative to those of non-PTSD participants, $t(56) = 2.24$, $p < .03$, whereas there were no significant group differences during the first two blocks.

Valence ratings. Analysis of the SAM valence ratings revealed a significant main effect of valence, $F(2, 54) = 130.29$, $p < .01$, with a significant linear trend, $F(1, 55) = 249.09$, $p < .001$, indicating greater valence ratings for pleasant than unpleasant pictures. There was also a main effect of block, $F(2, 54) = 4.98$, $p < .01$, with valence ratings tending to become less pleasant over the course of the procedure: baseline vs. posttrauma period comparison, $F(1, 55) = 4.20$, $p < .05$. Finally, the overall analysis also revealed a significant Block × Valence interaction, $F(4, 52) = 4.10$, $p < .01$. Decomposition of this effect indicated that the linear valence effect tended to dissipate from baseline to the posttrauma stressor period, $F(1, 55) = 5.27$, $p < .03$. There were no main or interactive effects of group.

Arousal ratings. Analysis of the SAM arousal ratings revealed a main effect of valence, $F(2, 54) = 64.38$ $p < .001$, with quadratic, $F(1, 55) = 131.09$, $p < .001$, and linear trends, $F(1, 55) = 10.09$, $p < .002$, indicating that arousal ratings were higher for pleasant and unpleasant images relative to neutral images and for unpleasant relative to pleasant pictures. There was also a marginal Block × Valence interaction, $F(4, 52) = 2.55$, $p < .05$, the decomposition of which revealed that the quadratic valence effect tended to dissipate over the course of the procedure: baseline vs. posttrauma challenge period, $F(1, 55) = 7.15$, $p < .01$. There were no main or interactive effects of group.

Discussion

This study examined the effects of trauma-related and non-trauma-related laboratory stressors on subsequent emotional behavior in combat veterans with and without PTSD. It was designed to test the hypothesis that emotional-processing abnormalities in PTSD occur secondary to the activation of trauma-related CERs and are reflected in hypo- and hyperresponsivity to pleasant and unpleasant stimuli, respectively. To do so, we conducted a replication and extension of a prior study by Litz et al. (2000) who found that individuals with PTSD exhibited suppressed zygomatic EMG (i.e., smile) responses to pleasant photographs after exposure to a trauma-related audiovisual stressor.

Affect-Modulated Startle and the Locus of Emotional-Processing Abnormalities in PTSD

One important addition to the procedures used in this study was the inclusion of startle reflex methodology to index emotional processing. Prior research has shown the eyeblink response to a startle-eliciting acoustic probe to be potentiated during viewing of unpleasant relative to pleasant pictures, a phenomenon that is believed to occur as a function of the match/mismatch between the strategic response disposition prompted by the visual foreground and the defensive reflex elicited by the noise probe (Lang et al., 1990). Following the hypothesis that emotional-processing abnormalities in PTSD occur primarily as a consequence of the activation of trauma-related CERs, we predicted state-specific alterations in emotion-modulated startle in individuals with PTSD. Specifically, we hypothesized that if the impact of the CER operates by mobilizing the defensive motive system at the strategic level, then we would expect to observe alterations in patterns of reflex modulation following presentation of a trauma-related stressor in participants with PTSD (i.e., less startle response inhibition and greater potentiation during viewing of pleasant and unpleasant pictures, respectively).

This hypothesis was partially supported. During the baseline interval prior to exposure to any laboratory stressor, startle responses in both groups were greater during viewing of unpleasant than pleasant images and were inhibited during picture viewing relative to ITIs (which suggests greater attentional engagement during picture processing; Codispoti et al., 2001; Simons & Zelson, 1985) irrespective of PTSD status. The two groups also exhibited equivalent baseline patterns of facial EMG response (i.e., increased zygomatic and corrugator activity during viewing of pleasant and unpleasant images, respectively), HR change (i.e., greater deceleration during viewing of unpleasant compared with pleasant pictures), and self-reported affective response.[3] These results (a) controvert the common conception that individuals with PTSD suffer from a generalized restriction in the capacity to experience emotion and (b) replicate findings by Litz et al. (2000) and Amdur, Larsen, and Liberzon (2000) who also observed no differences between participants with and without PTSD in patterns of emotional response to photographic stimuli under baseline or neutral conditions.

The two groups showed divergent patterns of startle modulation and facial EMG activity only after exposure to a trauma-related audiovisual stressor. Following this manipulation, individuals with PTSD exhibited significantly larger startle responses during viewing of unpleasant images and greater corrugator EMG activity irrespective of image valence relative to that exhibited by control participants. This finding is consistent with evidence that patterns of startle modulation are sensitive to negative mood manipulations, and that responses to aversive stimuli may be synergistically intensified under states of heightened defensive readiness in anxious individuals (Miller & Patrick, 2000). In contrast, the non-PTSD group showed no evidence of defensive priming. Instead, results for this group showed that picture valence effects tended to dissipate over the course of the procedure.

Lang et al. (1990) have proposed that the startle response taps the primary, strategic component of emotion, expressed along a continuum extending from appetitive to defensive action, with activation of the defensive system leading to potentiation of the blink reflex. From this standpoint, the finding of greater relative startle potentiation in the PTSD group following exposure to the trauma-related stressor suggests that this manipulation differentially activated the defensive system at the strategic level leading to priming of subsequent defensive emotional responses. The tactical expression of this defensive action disposition was reflected in generalized expressions of negative affectivity, indexed by corrugator EMG, that were discordant with the valence of the visual presentation. Thus, one interpretation of these findings is that trauma-related reexperiencing may engender a defensive response disposition that tends to color subsequent affective expressions irrespective of the valence of emotional cues present in the environment.

Contrary to prediction, however, there were no corresponding group differences in the degree of startle inhibition during viewing of pleasant pictures that might have suggested a phasic deficit in positive/appetitive motivation, nor did we replicate the finding of suppressed zygomatic EMG response to pleasant images after exposure to a trauma-related stressor in individuals with PTSD (Litz et al., 2000). Methodological differences between the two studies may have accounted for discrepancies in the patterns of results between the two studies. In designing this study, an extensive effort was undertaken to improve the quality of the trauma-related stressor—historically accurate and evocative war photos were obtained from a collection at the National Archives and digitized, resulting in a manipulation that was considerably more vivid and evocative than the one used in the first study (Litz et al., 2000).[4] The inclusion of two additional aversive laboratory procedures (i.e., startle recording and a shock manipulation) may have also contributed to the generally greater defensive activation observed in participants with PTSD in this study compared with that observed in the first.

Efficacy of the Stress Manipulations

A second extension from the Litz et al. (2000) study examined whether the impact of a trauma-related stressor on subsequent emotional behavior in PTSD is specific to the content of the manipulation (e.g., via activation of a network of trauma memories), or a more generalized consequence of heightened negative

[3] It is interesting to note that despite carefully selecting pleasant and unpleasant images with equivalent arousal ratings on the basis of the IAPS male college student-based norms (Lang, Bradley, & Cuthbert, 1999), analyses showed that both groups endorsed lower arousal ratings for pleasant compared with unpleasant images. This unexpected finding, suggesting a relatively restricted range in positive affective response in this sample, raises questions about how age and trauma exposure may influence the response to emotionally evocative stimuli, and should be addressed in future research.

[4] Analyses revealed that the trauma-related stressor in this study produced 10.1- and 4.4-point mean increases in PANAS–NA scores relative to postshock manipulation levels in the PTSD and non-PTSD groups, respectively. In comparison, unpublished results from our first study showed 5.6- and 1.2-point mean increases in NA as a function of the trauma challenge. Analysis of these data using a three-way repeated measures ANOVA that included study, group, and time (pre- vs. postprime) as factors revealed a significant Time $\times$ Study interaction, $F(1, 115) = 15.89$, $p < .01$, suggesting greater NA increases in response to the trauma-related challenge in this study compared with the NA increases found in the first study.

affectivity. Participants underwent trauma-related and non-trauma-related stressors with the prediction that alterations in emotional behavior would be evident only after the trauma-related manipulation. To test this hypothesis, we had two study design objectives: First, we aimed to implement a non-trauma-related stressor that would elicit comparable levels of negative affective arousal in participants with and without PTSD. The finding that the two groups produced equivalent startle, HR, SCL, corrugator, and PANAS–NA increases in response to the shock manipulation suggests that this objective was met.[5] Second, we aimed to develop trauma-related and non-trauma-related stressors that would elicit comparable levels of reactivity in the non-PTSD group. Results showed that although non-PTSD participants showed equivalent HR, SCL, and corrugator increases during exposure to the two stressors, both groups endorsed greater distress in relation to the trauma manipulation compared with the shock manipulation.[6] The finding of greater self-reported distress in the non-PTSD group in response to the trauma stressor compared with the shock challenge was unexpected and may have reflected the shared learning history of the two groups and the sheer intensity of the trauma-related-stress manipulation.

Finally, our prediction that participants with PTSD would show greater negative emotional reactivity during presentation of the trauma-related audiovisual stimulus was supported by evidence of robust increases in HR and self-reported negative affect in the PTSD group. Replicating results of our first study, however, we found no significant differences between groups in the magnitude of corrugator EMG and SCL change in response to this stressor. As others have noted (Blanchard, Hickling, Buckley, Taylor, Vollmer, & Loos, 1996), HR increase may be a uniquely sensitive marker of PTSD-related reactivity to trauma-related stimuli.

Impact of the Trauma Challenge on Perceptual Engagement

A novel finding from the startle response component of this study was that the two groups differed following exposure to the trauma-related challenge, not only with respect to the magnitude of blink responses during viewing of unpleasant images, but also in terms of the degree to which startle responses were inhibited during picture processing relative to no-picture trials (ITIs). During picture processing, blink responses to acoustic probes are generally inhibited relative to no-picture trials, as was the case in this study overall. This is believed to occur because the allocation of attentional resources to the processing of stimuli in a given modality results in an inhibition of processing input from other modalities (Anthony, 1985). Thus, the difference in acoustic startle amplitude between picture and no-picture trials is thought to provide an index of the degree of perceptual engagement in processing of the visual foreground stimulus (Anthony, 1985; Simons & Zelson, 1985). The pattern of results observed during the third block of our study, however, suggests a significant reduction in the degree of engagement in processing of the visual foreground stimulus in participants with PTSD. This generally fits with theories that have proposed that disruptions of, or disengagement from, the processing of sensory stimuli may occur in response to activation of a trauma-related CER (Pitman, van der Kolk, Orr, & Greenberg, 1990; Spiegel, 1997).

Methodological Limitations

There are several more general methodological limitations to this study that should be considered as well. First, group differences cannot be solely attributed to PTSD status because there were substantial differences between groups in terms of medication status, alcohol and drug problems, and depression. Second, the non-trauma-related and trauma-related stressors were presented in a fixed order that confounded habituation effects with stressor type. However, although this may have resulted in a general attenuation of picture valence effects over the course of the procedure, there is little reason to believe that the two groups would exhibit differential rates of habituation of picture valence effects. Third, although the non-trauma-related (shock) stressor was effective in evoking acute increases in physiological arousal, the PANAS–NA data suggest that the manipulation had a limited impact on self-reported mood state. Fourth, though the trauma-related stressor involved actual exposure to an aversive audiovisual stimulus, the threat of shock involved anticipation of an aversive tactile stimulus. Thus, in future research along these lines it would be beneficial to use manipulations that are equivalent in terms of presentation condition (anticipation vs. exposure), modality (i.e., audiovisual only or perhaps imagery only), and efficacy in evoking sustained negative arousal effects.

Conclusions and Implications for Future Research

The results of this study are consistent with the theory that abnormalities in emotional behavior in PTSD are largely state-dependent and may occur in circumstances involving the activation of trauma-related CERs (Litz, 1992). In this study, individuals with PTSD showed a priming of defensive emotional responses and behavior indicative of perceptual disengagement, only after being exposed to reminders of their trauma. Similarly, Litz et al. (2000) observed emotional-processing abnormalities in partici-

[5] It is noteworthy that the finding of comparable levels of startle potentiation in PTSD and non-PTSD groups in response to the shock threat replicates, in a somewhat different paradigm, findings of prior research (e.g., Grillon, Morgan, Davis, & Southwick, 1998; Morgan, Grillon, Southwick, Davis, & Charney, 1995). Unlike those investigators, however, we found no significant group differences in baseline startle amplitude in a context involving aversive anticipation.

[6] Another unexpected finding associated with the trauma challenge was that startle responses were attenuated during exposure to trauma-related stimuli compared with baseline. In light of the fact that both groups exhibited robust startle potentiation during the shock manipulation, and that both groups endorsed greater NA in response to the trauma challenge compared with the shock threat, this finding suggests that the potentiating effects of heightened NA on startle amplitude were superceded by the impact of greater attentional engagement in processing of the visual foreground stimulus which inhibits startle. Indeed, the two manipulations differed substantially in terms of the visual stimuli. During the non-trauma-related stressor the word *shock* was the only visual stimulus presented, whereas during the trauma-related challenge 30 evocative photographs of war were presented in a series. Despite the fact that startle probes were presented during 2-s intervals between the picture presentations, perceptual processing resources during the trauma challenge were likely to have been allocated to a greater degree to the visual modality resulting in attenuation of the responses to acoustic probe stimuli.

pants with PTSD only after exposure to a similar manipulation. Neither study found evidence of a trait-like deficit in emotionality or a general suppression of emotional responsiveness associated with PTSD that is implied by the *DSM–IV* definition of the disorder. Accordingly, a reevaluation of the symptom class of emotional numbing in PTSD may be warranted.

The findings of this study suggest that the emotional-processing abnormalities of individuals with PTSD are characterized, at least in part, by enhanced negative affective reactions to trauma-related cues that prime defensive responding to other negatively valent (but not necessarily trauma-related) stimuli. They also are consistent with Litz's (1992) hypothesis that when individuals with PTSD are cued by trauma-related contexts they are more responsive to emotional stimuli that are affectively consistent with their elicited state. It remains unclear, however, whether negative emotional responses are more readily activated in individuals with PTSD, if priming effects are more persistent in these individuals, or both. Whereas the former would suggest a heightened sensitivity of the defensive emotional system, the latter would suggest a deficit in affect regulation involving the capacity to recover from reexperiencing states. These alternative hypotheses may be fruitful avenues for future research on the emotional-processing abnormalities of individuals with PTSD.

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